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Health and Environmental Effects Profile for Bromozynil

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR BROMOXYNIL

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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16. ABSTRACT

The Health and Environmental Effects Profile for bromoxynil was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Bromoxynil has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for bromoxynil is 6×10^{-2} (mg/kg bw/day) for oral exposure.

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This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

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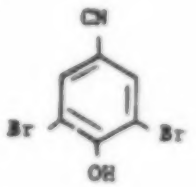
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
NOEL	No-observed-effect level
ppm	Parts per million

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Bromoxynil is the common name for 3,5-dibromo-4-hydroxybenzonitrile. It is also known as 3,5-dibromo-4-hydroxyphenyl cyanide (BCPC, 1977) and 4-hydroxy-3,5-dibromo-benzonitrile, brittox, brominal, brominen, broxynil and buctril (NIOSH, 1983). In the USSR, it is known as butilchlorofis (Wiswesser, 1976). This compound was introduced in 1963 by Amchem Products, Inc. under the trademark "Brominil" and by May and Baker Ltd. under the code number "MB 10,064" and the trademark 'Buctril', (BCPC, 1977). The structure of bromoxynil is depicted below:



Molecular formula: $C_7H_3Br_2NO$

Molecular weight: 276.93

The Chemical Abstracts Service (CAS) Registry Number for this compound is: 1689-84-5.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Bromoxynil is a white, odorless solid. Some of the physical properties are listed below (BCPC, 1977):

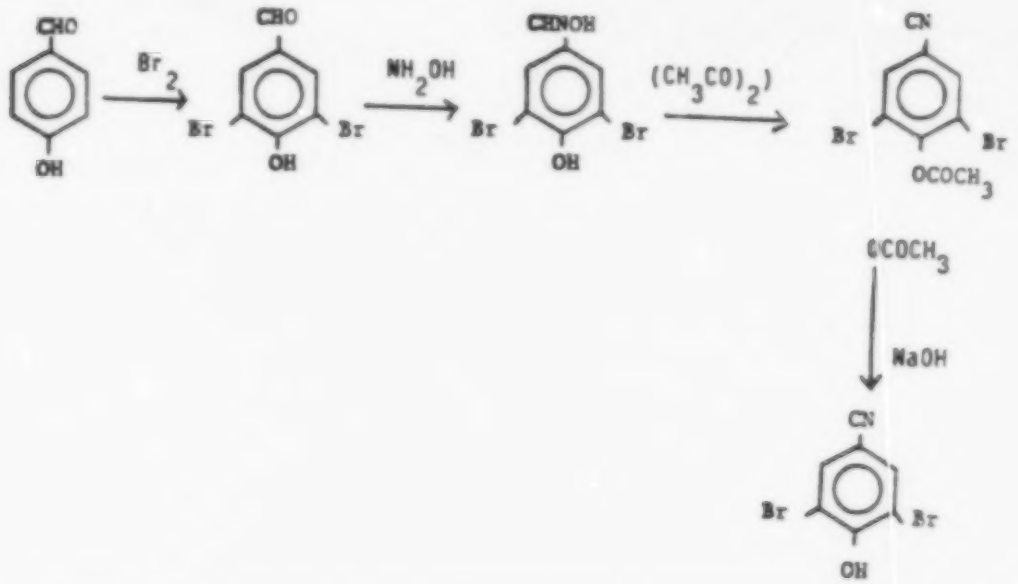
Melting point:	194-195°C
Water solubility at 25°C:	130 mg/ℓ
Solubility in acetone:	170 g/ℓ
methanol:	90 g/ℓ
petroleum oils:	<20 g/ℓ
Sublimes at 135°C at 0.15 torr Vapor pressure:	Negligible vapor pressure up to 35°C

Bromoxynil forms water-soluble alkali metal and amine salts that are stable and noncorrosive.

Bromoxynil has a pK_a of 4.06 (Windholz, 1983).

1.3. PRODUCTION DATA

The synthesis of bromoxynil begins with p-hydroxybenzaldehyde, as follows (Plimmer, 1970a):



The following are some of the manufacturers of bromoxynil:

Producer	Location	Reference
Rhone-Poulenc, Inc. Agrichemical Division	Portland, OR	SRI, 1983
Rhone-Poulenc, Inc. Agriculture Division	Monmouth Junction, NJ	U.S. EPA-NIH, 1983
Union Carbide Corp. Agricultural Products Group	Institute, WV	SRI, 1983
Union Carbide Corp. Agricultural Products Co., Inc.	Ambler, PA	U.S. EPA-NIH, 1983

Information on production volumes of bromoxynil, per se, was not located; however, the 1977 TSCA Production File lists Union Carbide as a producer of the octanoate formulation of bromoxynil at a production volume of 100,000-1,000,000 pounds annually.

1.4. USE DATA

Bromoxynil is a herbicide used to control weeds in wheat, barley, oat, rye, flax, grass for sod and noncrop areas (Berg, 1980).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

The two likely routes of entry of this herbicide into the atmosphere are spray drift during application of the herbicide to soil and evaporation from the soil after application. No information pertaining to the fate or transport of this chemical in the atmosphere was found in the available literature. The fate of bromoxynil in the air is likely to be determined by its ability to undergo reactions with O_3, NO_x and free radicals (i.e., •OH) and by its ability to undergo photochemical reactions. In the absence of any qualitative or quantitative data pertaining to such reactions, it is difficult to predict the atmospheric fate of this compound.

No studies relating to the transport of bromoxynil in the atmosphere were found in the available literature; it is difficult to make theoretical predictions based on the limited amount of data.

Little information pertaining to the fate and transport of bromoxynil in water were found in the available literature. Chemically, bromoxynil could react with the available free radicals in the aquatic media or undergo photochemical and hydrolytic reactions. No data were available regarding the reactivity of bromoxynil with free radicals. Hsu and Camper (1975) reported that bromoxynil could undergo hydrolysis in basic solution to its amide and acid derivatives. In aqueous solutions, bromoxynil was decomposed by light to polyphenols and colored polymeric materials; however no rates were discussed (Plimmer, 1970b). No specific data were encountered regarding the significance of microbial degradation in the aqueous media, although some information can be obtained from soil degradation studies. Based on its biodegradability in soils (Smith, 1971; Smith and Cullimore, 1974; Cullimore and Kohout, 1974; Hsu and Camper, 1975), it is likely that biodeg-

radation will play an important role in determining the fate of this compound in water.

Specific data regarding the aquatic transport of bromoxynil by the processes of volatilization or sorption were not found in the available literature; however, some predictions can be made based on the physical properties of this compound. Based on the neglible vapor pressure of up to 35°C (BCPC, 1977), a moderate water solubility (130 mg/ℓ) and a sorption coefficient of 300 calculated from its water solubility (Kenaga, 1980), it is unlikely that bromoxynil will volatilize readily from water.

No experimental data regarding the bioconcentration of bromoxynil by aquatic organisms were available. Based on a calculated bioconcentration factor of 40 (Kenaga, 1980), bromoxynil is not expected to bioaccumulate significantly in aquatic organisms.

Limited data on the fate of bromoxynil in soil were available. No information regarding the significance of chemical or photochemical reactions was found in the available literature. Microbial degradation of bromoxynil, however, has been studied. Smith (1971) reported that bromoxynil was rapidly degraded to an amide in moist, nonsterile Regina heavy clay soil, with breakdown occurring more rapidly at 25°C than at 18°C. The half-life of the herbicide was <2 weeks under field conditions. In a later study, Smith (1980) reported that following the degradation of bromoxynil octanoate to bromoxynil in heavy clay or sandy loam at 20°C, the further degradation of bromoxynil was 30% complete at 24 hours and 96-97% complete at 7 days. Bromoxynil was found to have a half-life of ~7 days in a clay loam soil under laboratory conditions (Hsu and Camper, 1975). Smith and Cullimore (1974) concluded that bromoxynil underwent microbial hydrolysis in the presence of a flexibacterium strain BR4 to the corresponding amide and

that the amide was further hydrolyzed to the acid analogue. Cullimore and Kohout (1974) studied the microbial degradation of bromoxynil both in pure culture and in soil and found rapid degradation in soil (14 days at 25°C based on a bioassay). It is likely, therefore, that microbial degradation will play a major role in the fate of this compound in the soil.

Information regarding the transport of bromoxynil in the soil was not found in the available literature. The two processes that may influence the transport of this compound are leaching and volatilization. Both of these processes are controlled to some extent by the adsorptive capability of bromoxynil to soil particles. This compound has a moderate adsorption coefficient of 300, calculated from its water solubility (Kenaga, 1980); therefore, it may sorb to soil particles. Given its low K_{oc}, bromoxynil may leach despite its relatively low water solubility.

3. EXPOSURE

Monitoring data for this compound were not located in STORET or elsewhere.

4. PHARMACOKINETICS

The fate of bromoxynil has been studied in lactating cows that were fed dietary levels of 5 ppm for 4 days. No residues was detected in milk or feces and ~19% of the administered dose was excreted in the urine as the parent compound or conjugates of the parent compound (St. John and Lisk, 1967). No other information regarding the absorption, distribution, metabolism or excretion of this compound was located in the available literature.

5. EFFECTS

Limited information regarding the mutagenicity, teratogenicity, reproductive toxicity, and subchronic testing was found in a Federal Register article on tolerances for bromoxynil residues. Because no details were provided, this information is presented only in Section 7.1.

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of bromoxynil were not located in the available literature. This compound is not scheduled for testing by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

Limited information on the mutagenic potential of bromoxynil was available. Bromoxynil failed to induce mutations in the plant Pelargonium zonale when one drop at a 10^{-3} M solution was added to the terminal bud, and the leaves that subsequently developed were observed for chlorophyll defects and morphological changes (Pohlheim et al., 1977). Negative results were obtained in a mutagenicity assay with eight histidine-requiring strains of S. typhimurium tested in the absence of an S-9 activating fraction (Anderson et al., 1972). The assay was performed as a spot test, with a small crystal of bromoxynil added to the plate. These limited data are inadequate to conclude that bromoxynil is not mutagenically active.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of bromoxynil were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of bromoxynil were not located in the available literature.

5.5. CHRONIC TOXICITY

Pertinent data regarding the chronic toxicity of bromoxynil were not located in the available literature.

5.6. OTHER RELEVANT INFORMATION

Oral LD_{50}s were reported as 190 mg/kg for the rat, 110 mg/kg for the mouse, 260 mg/kg for the rabbit, and 63 mg/kg for the guinea pig. An intravenous LD_{50} for the mouse was 56 mg/kg (NIOSH, 1983).

6. AQUATIC TOXICITY

6.1. ACUTE TOXICITY

Changes in the pH of water affect the proportion of bromoxynil that is present in the toxic un-ionized phenol form. The pK_a of bromoxynil is 4.06 (Windholz, 1983). In acidic water where the phenol form predominates, bromoxynil is more toxic than in basic water where the ammonium salt forms (Grohmann and Sobhani, 1980). For example, the LC_{50} values of bromoxynil to fish (species and exposure period not specified) were 0.2, 2.0 and 20.0 mg/ℓ in water of pH 6.2, 7.2 and 8.2, respectively (Grohmann and Sobhani, 1978). The 24- and 48-hour LC_{50} values of bromoxynil (potassium salt) in hard water (250 mg/ℓ as $CaCO_3$) for the harlequin fish, Rasbora heteromorpha, were 63.0 and 60.0 mg/ℓ, respectively (Alabaster, 1969). In soft water (20 mg/ℓ as $CaCO_3$) the 24- and 48-hour LC_{50} values were 5.2 and 5.0 mg/ℓ, respectively. Although it was stated that the pH was ~7.2, the hardness may have altered the pH enough to cause the 10-fold change in lethality levels (Alabaster, 1969; Grohmann and Sobhani, 1978).

6.2. CHRONIC EFFECTS

Pertinent data regarding the effects of chronic exposure to bromoxynil were not located in the available literature.

6.3. PLANT EFFECTS

Bromoxynil at 10 mg/ℓ caused at least 50% inhibition of growth in the green algae, Chlorella, detected by chlorophyll content after 18-36 hours incubation (Kratky and Warren, 1971). At 1 mg/ℓ, bromoxynil did not cause a 50% reduction of growth. Cullimore (1975) reported that bromoxynil at concentrations up to 10 mg/ℓ did not cause a 50% reduction of cell growth in 16 species of algae (including Chlamydomonas, Chlorella, Coccomyxa, Haematococcus, Hormidium, Mesotaenium, Scenedesmus, Spongiochloris and

Stichococcus sp.). Growth was reduced at least 50% in Hormidium barlowi at 0.5-2 mg bromoxynil/ℓ and inhibited completely at 5.0 mg/ℓ (Cullimore, 1975). Fletcher et al. (1970) reported that bromoxynil (concentration not reported) was algistatic in Chlarmydomonas globosa, Chlorella pyrenoidosa and Stichococcus bacillaris, and the effect was strongly influenced by pH.

6.4. RESIDUE

Pertinent data regarding bromoxynil residues in aquatic organisms were not located in the available literature.

6.5. OTHER RELEVANT INFORMATION

Additional data regarding the effects of bromoxynil on aquatic organisms or in aquatic environments were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

In 1982, in response to petitions of Union Carbide Agricultural Products Co., Inc. and Rhone-Poulenc, Inc., the U.S. EPA (Federal Register, 1982a,b), respectively established a maximum permissible level of 0.1 ppm for residues of the herbicide bromoxynil resulting from the application of bromoxynil octanoate (and bromoxynil butyrate) in or on raw agricultural commodities. Since the tolerance level was established for residues of bromoxynil, rather than for bromoxynil octanoate (or butyrate), it is assumed that the toxicological data on which the level was based apply to bromoxynil, rather than to the octanoate (or the butyrate), although this was not clear from the two accounts (Federal Register, 1982a,b). The data included a 13-week feeding study in the dog with a NOEL of 5 mg/kg/day, a 13-week feeding study in the rat with a dietary NOEL of 312 ppm, a 3-generation dietary study in the rat with a NOEL of $\geq$300 ppm and a negative mutagenicity assay in S. typhimurium (Federal Register, 1982a,b). The maximum permissible level was also based on the results of a teratogenicity study (or studies) in rats. A NOEL for teratogenic effects of 15 mg/kg/day was reported in one account (Federal Register, 1982a), but a NOEL of >35 mg/kg for teratogenicity with fetotoxicity at >5 mg/kg was reported in the later account (Federal Register, 1982b). The reason for the discrepancy in teratogenicity NOELs between the accounts is not apparent. Further information regarding any of these toxicological studies was not provided by U.S. EPA (Federal Register, 1982a,b) and these studies were not encountered in the available published literature. Based on the 13-week study in dogs and using an uncertainty factor of 2000, an ADI for bromoxynil of 0.0025 mg/kg/day was calculated (Federal Register, 1982a,b).

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of bromoxynil were not located in the available literature.

8. RISK ASSESSMENT

No carcinogenicity data for bromoxynil were located in the available literature. The available mutagenicity data are inadequate for evaluating the mutagenicity of bromoxynil.

No studies regarding the effects of subchronic or chronic exposure to bromoxynil were located in the published literature. Unpublished oral teratogenicity and reproductive effects studies (rat) and unpublished subchronic feeding studies (dog and rat) were mentioned briefly in Federal Register articles that set tolerances for bromoxynil on raw agricultural commodities (Federal Register, 1982a,c). Using a safety factor of 2000, an ADI of 0.0025 mg/kg/day (equivalent to 0.175 mg/day for a 70 kg man) was calculated from a NOEL of 5 mg/kg/day in the subchronic study with dogs (Federal Register, 1982a,c). The adequacy of the studies, and consequently of this ADI, could not be evaluated because no details regarding the studies were available in the Federal Register accounts.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

The CBI Appendix to this document reviews unpublished animal studies; however, the subchronic feeding study in dogs noted in the Federal Register (1982a,c) was not among the studies available for bromoxynil. As discussed

in the profile on bromoxynil octanoate (U.S. EPA, 1984), a subchronic study in dogs fed bromoxynil octanoate was available and may be the study mentioned in the Federal Register (1982a,c).

The CBI Appendix also reviews chronic feeding studies of bromoxynil in rats and mice, which provide more appropriate data from which to derive an ADI. Based upon a NOAEL of 5.9 mg/kg bw/day for increased absolute liver weight in rats in a chronic feeding study and using an uncertainty factor of 100, an ADI of 0.06 mg/kg bw/day or 4.2 mg/day for a 70 kg man is recommended.

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Wiswesser, W.J., Ed. 1976. Pesticide Index, 5th ed. Entomological Society of America, College Park, MD. p. 30.

APPENDIX:

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. TLVs: Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981). Cincinnati, OH. p. 486.

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